SCIENTIFIC LETTER

Serum cardiac troponin T and plasma brain natriuretic peptide in patients with cardiac decompensation

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In our earlier study, idiopathic dilated cardiomyopathy patients with persistently high serum troponin T (TnT) concentrations had notably depressed left ventricular ejection fractions, increased left ventricular diastolic dimensions, and adverse long term outcomes.[1–3] Therefore, TnT seems to be a marker of ongoing subclinical myocyte injury, although the mechanism of its release is unknown. Most patients with poor outcomes had persistently high TnT concentrations, even when clinically stabilised by conventional treatment, when they were free of dyspnoea, or radiographic and auscultatory signs of pulmonary congestion. Brain natriuretic peptide (BNP) is an amino acid peptide secreted chiefly by the ventricular myocardium in response to strain. Measurements of plasma concentrations of BNP are being increasingly used as a clinical diagnostic, prognostic, and monitoring tool in patients with congestive heart failure (CHF).[4 5] We therefore examined the significance of TnT and BNP concentrations in patients with CHF.

METHODS

The study population comprised 35 consecutive patients who presented to the Hyogo Prefectural Amagasaki Hospital with CHF and pulmonary congestion between April 2000 and June 2001 (19 men and 16 women, mean age 67.8 years). Clinical diagnosis was idiopathic dilated cardiomyopathy in 16 patients, old myocardial infarction in 10 patients, valvar disease in 6 patients, hypertensive heart disease in 1 patient, and atrial fibrillation in 2 patients. No patient had a recent myocardial infarction or unstable angina pectoris within one year, and no electrocardiographic changes or increases in creatine kinase enzyme were observed throughout the study period. The criteria for a diagnosis of left heart decompensation used in this study consisted of the following findings on initial presentation: (1) dyspnoea or orthopnoea requiring emergent hospitalisation, intravenous frusemide (furosemide), and infusion of nitrates or inotropic agents; (2) radiographically apparent pulmonary oedema and presence of auscultatory moist rales. TnT $\geq$ 0.02 ng/ml was considered as significant.[3]

RESULTS

All patients had increased plasma baseline BNP concentrations (mean (SD) 927.3 (672.0) pg/ml, range 105–3121 pg/ml) at entry, while simultaneously measured serum TnT concentrations were increased to a mean of 0.054 (0.041) ng/ml in 22 patients (63%, group 1), versus < 0.02 ng/ml in 13 patients (group 2) at baseline with pulmonary congestion. BNP concentrations in patients in group 1 were significantly higher than in patients in group 2 (1114.3 (708.4) pg/ml *v* 610.8 (477.9) pg/ml, $p < 0.05$). Pulmonary congestion resolved in all patients after intravenous treatment of CHF. Although BNP concentrations significantly decreased after treatment in all patients to 385.3 (370.4) pg/ml (range 21–1480 pg/ml, $p < 0.0001$ versus baseline, $n = 35$), post-treatment BNP concentrations in group 1 patients remained significantly higher than in group 2 patients (510.5 (397.5) pg/ml *v* 183.2 (205.8) pg/ml, $p < 0.05$). TnT remained increased at a mean of 0.046 (0.031) ng/ml (range 0.02–0.14 ng/ml) in 20 patients in group 1, despite radiographic resolution of pulmonary congestion after a mean treatment period of 14.3 (13.3) days (range 5–50 days). During long term follow up, 13 of 22 patients in group 1 were rehospitalised for management of cardiac decompensation, 7 of whom ultimately died. In contrast, a single patient required rehospitalisation in group 2, all of whom survived. The event-free survival in group 1 was significantly lower than in group 2 ($p < 0.005$).

DISCUSSION

To identify potential differences between TnT and BNP release, we deliberately selected a patient population presenting with decompensated CHF and pulmonary congestion. Whereas baseline plasma concentrations of BNP were increased in all patients, initial TnT concentrations were < 0.02 ng/ml in approximately a third of patients, suggesting that, in this kind of population, an increase in TnT is not a marker of decompensated CHF. Patients with initially increased serum TnT concentrations had higher BNP concentrations than patients with low baseline TnT, including after resolution of pulmonary congestion by intravenous treatment. Ultimately, these patients generally continued to have increased TnT concentrations and had unfavourable outcomes, in contrast to patients with initially low TnT concentrations, whose outcomes were favourable. We hypothesise that an increase in TnT concentrations is an expression of ongoing myocyte injury unmitigated by treatment of CHF, and associated with a greater rise in BNP concentrations.

Our limited patient population did not enable the identification of interventions which effectively lower serum TnT concentrations. Large clinical drug trials monitoring these parameters are necessary. BNP and TnT are both easy to measure, require simple laboratory methods, and may be repeated many times to follow patients, without interobserver variability. Theoretically, BNP is a marker of heart failure status and TnT is a marker of myocyte injury. The first therapeutic goal may be relief of circulatory congestion and lowering of BNP. The second goal may be attenuation of myocyte injury and lowering of TnT. When all conventional efforts have failed to decrease TnT and BNP, more aggressive steps, including cardiac transplantation, might be considered.

Abbreviations: BNP, brain natriuretic peptide; CHF, congestive heart failure; TnT, troponin T

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IMAGES IN CARDIOLOGY

Successful use of the Guardwire Plus distal embolisation protection device in a native left anterior descending coronary artery

A 66 year old man was transferred for coronary angiography 48 hours after presentation to a referring hospital with an acute coronary syndrome. Selective injection of the left coronary artery revealed a large mobile thrombus in the proximal left anterior descending artery (panel A, arrow). Removal of the thrombus with the Possis AngioJet rheolytic thrombectomy catheter was considered, but was rejected in view of the mobility of the thrombus and the risk of embolisation. Instead, the Medtronic Guardwire Plus system, a distal embolisation protection device, was used. This device has been evaluated in saphenous vein graft interventions but has not been tested for use in native coronary arteries, nor has it been approved by the US Food and Drug Administration. The system consists of a low profile elastomeric occlusion balloon at the end of a specialised 0.014 inch, nitinol, hypotube angioplasty wire. Abciximab was commenced and the lesion was crossed with the Guardwire. The occlusion balloon was inflated distal to the lesion resulting in temporary obstruction (panel B, arrow). Using the Guardwire the lesion was directly stented with a 4×12 Nir Elite stent during distal occlusion (panel C). The stent balloon was removed and the Export aspiration catheter passed into the occluded artery through which 20 ml of blood and thrombotic debris was aspirated. The distal occlusion balloon was then deflated restoring antegrade flow (panel D). Subsequent angiography showed TIMI 3 flow, good myocardial contrast blush, and no obvious loss of distal branches. Post-procedural enzymes were not raised suggesting little distal embolisation of thrombus.

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